1B-2, and 1B-3 show the nucleic acid and amino acid sequence of SEQ ID Nos. 2 and 5, respectively. Figure 1D-1 to 1D-3 show the nucleic acid and amino acid sequence of SEQ ID Nos. 3 and 6, respectively.—

-- Figure 2 represents the amino acids sequence of the active human CCR5 chemokine receptor (SEQ ID NO: 5) according to the invention aligned with that of the human CCR1 (SEQ ID NO: 9), CCR2b (SEQ ID NO: 7), CCR3 (SEQ ID NO: 8), and CCR4 (SEQ ID NO: 10) receptors. Amino acids identical with the active CCR5 sequence are boxed. --

-- Figure 6 represents the structure of the mutant form of human CCR5 receptor. Figure 6A shows a diagram of the mutant form of human CCR5 receptor (SEQ ID NO:18) situated in a membrane. Figure 6B shows the wild type amino acid sequence (SEQ ID NO: 11), and the location of the 32 base deletion mutation in the nucleic acid (SEQ ID NO: 12) and amino acid sequences (SEQ ID NO: 13). –

On page 1, please replace the first paragraph under the heading "BACKGROUND", and extending from lines 4-5 with the following paragraph.

-- This application is a divisional of U.S. Patent Application Serial No. 09/626,939, filed July 27, 2000, now abandoned, which is a continuation of U.S. Patent Application Serial No. 08/833,752, filed April 9, 1997, now U.S. Patent No. 6,448,395, which claims priority under 35 U.S.C. § 119(a)-(d) to EP 96870021.1, filed March 1, 1996, and EP 96870102.9, filed August 6, 1996.--

On page 29, please replace the paragraph, extending from lines 1-23, with the following replacement paragraph;

-- seronegative individuals as controls; the full coding region of their CCR5 gene was amplified by PCR and sequenced. Unexpectedly, one of the slow progressors, but also two of the uninfected controls, exhibited heterozygosity at the CCR5 locus for a biallelic polymorphism. The frequent allele corresponded to the published CCR5 sequence, while the minor one displayed a 32 bp deletion within the coding sequence, in a region corresponding to the second extracellular loop of the receptor (Fig. 6). The figure 6 is

the structure of the mutant form of human CC-chemokine receptor 5. *a*, The amino acid sequence of the non-functional Δccr5 protein is represented. The transmembrane organisation is given by analogy with the predicted transmembrane structure of the wild-type CCR5. Amino acids represented in black correspond to unnatural residues resulting from the frame shift caused by the deletion. The mutant protein lacks the last three transmembrane segments of CCR5, as well as the regions involved in G protein-coupling *b*, Nucleotide sequence of the CCR5 gene surrounding the deleted region, and translation into the normal receptor (top) or the truncated mutant (ccr5, bottom). The 10-bp direct repeat is represented in italics. The full size coding region of the CCR5 gene was amplified by PCR, using 5'-TCGAGGATCCAAGATGGATTATCAAGT-3' (SEQ ID NO: 14) and 5'-CTGATCTAGAGCCATGTGCACAACTCT-3' (SEQ ID NO: 15) as forward and reverse primers' respectively. The PCR products were sequenced on both strands using the same oligonucleotides as primers, as well as internal primers, and fluorochrome-labelled dideoxynucleotides as terminators. The sequencing products were run on an Applied Biosystem sequencer, and ambiguous positions were searched along the coding sequence. When the presence of a deletion was suspected from direct sequencing, the PCR products were cloned after restriction with *BamHI* and *XbaI* endonucleases into pcdna3. Several clones were sequenced to confirm the deletion. The deletion was identical in three unrelated individuals investigated by sequencing.--

On page 32, please replace paragraph 1 extending from lines 1-9 with the following replacement paragraph;

-- CCTGGCTGTCGTCCATGCTG-3' (SEQ ID NO: 16) and 5'-CTGATCTAGAGCCATGTGCACAACTCT-3' (SEQ ID NO: 17) as forward and reverse primers respectively. Reaction mixtures consisted in 30 µl of 10 Mm Tris-HCl buffer pH 8.0, containing 50 Mm KCl, 0.75 Mm $MgCl_2$, 0.2 Mm dCTP, dGTP and dTTP, 0.1 Mm dATP, 0.5 µi [a-^{32}P]-dATP, 0.01% gelatin, 5% DMZ, 200 ng target DNA, 60 ng of each of the primers and 1.5 U Taq polymerase. PCR conditions were: 93 °C for 2 min 30; 93 °C for 1 min, 60 °C for 1 min, 72 °C for 1 min, 30 cycles; 72 °C for 6 min. After the PCR reaction, the samples were incubated for 60 min at 37 °C with 10 U